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The Effect of Lead on Erythrocyte Glucose-6-Phosphate Dehydrogenase Activity in Rats

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Background: Exposure to lead damages the biological systems, developing oxidative stress. Glucose-6-phosphate dehydrogenase (G6PD) produces NADPH in pentose phosphate pathway. This molecule protects tissues from oxidative stress. There are conflicting reports in the literatures of the effects of lead on G6PD activity.

Objectives: The aim of this investigation was to evaluate the effect of lead on erythrocyte G6PD activity in rats given lead acetate in their drinking water.

Materials and Methods: In this study 14 albino rats were divided into two groups of seven animals. The treated group was exposed to 2% lead acetate in the drinking water during eight weeks. The control group was kept in the similar condition as the test group; however this group was not exposed to lead acetate. The blood lead level was measured with an atomic absorption spectrophotometer. The G6PD activity was determined by kinetic method. The hemoglobin content was determined by Drabkin's method. Malondialdehyde (MDA) in rats' plasma was measured with the thiobarbituric acid test using HPLC.

Results: The blood lead concentration in treated group was increased when compared to control group ($P < 0.05$). A significant decrease in hemoglobin level was noted in lead-treated animals ($P < 0.05$). The G6PD activity in erythrocytes of rats received lead acetate increased up to 81% compared to the control group ($P < 0.05$). The G6PD/hemoglobin ratio in control and treatment groups was 17.7 ± 3.6 U/g and 44.9 ± 4.4 U/g, respectively. A significant increase in the plasma MDA level was also observed in the exposed group ($P < 0.05$).

Conclusions: The results of this study showed that exposure to lead increased the activity of G6PD in the rat erythrocytes, perhaps resulting in an up regulation of the enzyme to detoxify lead.

Keywords: Glucose-6-Phosphate Dehydrogenase; Malondialdehyde; Lead; Oxidative Stress; Rats

1. Background

Lead is a toxic heavy metal which damages the biological systems. The toxic effect of lead and its compounds in animals and humans have been investigated for many years. Exposure to lead is associated with a number of morphological, physiological and biochemical abnormalities. Lead toxicity was observed in acute or chronic forms and its magnitude depends on many parameters. It can accumulate in some organs and results in kidney failure and abdominal pain. Also, lead diffuses in bone matrix and replaces calcium. Lead toxicity especially in children damages the nerve system and causes a number of behavioral abnormalities. It inhibits delta-aminolevulinic acid (ALA) dehydratase and ferrochelatase enzymes in the heme biosynthesis pathway and causes blood disease (1, 2). Several mechanisms have been suggested for lead toxicity on biological systems (3, 4). Lead is mainly located in erythrocytes in blood and only 1% of lead is remained in serum. Recent studies have proposed that lead is capable of causing oxidative stress in erythrocytes. It induces an imbalance between generation and removal of reactive oxygen species (ROS) in cells and tissues that it causes damage to DNA, proteins and membranes. It is suggested that lead affects the activity of enzymes responsible for maintenance of redox balance in organisms (5, 6).

G6PD (EC 1.1.1.49) is the first enzyme in the pentose phosphate metabolic pathway. This enzyme supplies cells with most of the NADPH through the oxidation of

Implication for health policy/practice/research/medical education:
From the data presented in the current study, it appears that exposure to lead could increase the activity of G6PD and G6PD/hemoglobin ratio in the rat erythrocytes in vivo.